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=> fil capl; d que l11; fil medl; d que l27;fil biosis; d que l39; fil wpids; d que l40
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FILE COVERS 1967 - 14 Oct 1999  VOL 131 ISS 16
FILE LAST UPDATED: 13 Oct 1999  (19991013/ED)
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L2          2881 SEA FILE=CAPLUS ABB=ON  ALPHA 7
L5          1413 SEA FILE=CAPLUS ABB=ON  NICOTINIC RECEPTORS/CT
L6           106 SEA FILE=CAPLUS ABB=ON  L5(L)L2
L7        429676 SEA FILE=CAPLUS ABB=ON  ?NUCLEIC
L9         22646 SEA FILE=CAPLUS ABB=ON  DNA SEQUENCE#/CT
L10       372433 SEA FILE=CAPLUS ABB=ON  GENE#/CW
L11           17 SEA FILE=CAPLUS ABB=ON  L6 AND (L7 OR L9 OR L10)

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 FILE LAST UPDATED: 12 OCT 1999 (19991012/UP). FILE COVERS 1960 TO DATE.
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L12         5052 SEA FILE=MEDLINE ABB=ON  RECEPTORS, NICOTINIC/CT
L13         1031 SEA FILE=MEDLINE ABB=ON  ALPHA 7
L15           62 SEA FILE=MEDLINE ABB=ON  BASE SEQUENCE+NT/CT AND L13
L21         1012 SEA FILE=MEDLINE ABB=ON  L12 AND HUMAN/CT
L27            8 SEA FILE=MEDLINE ABB=ON  L21 AND L15

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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 8 October 1999 (19991008/ED)

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L29       551162 SEA FILE=BIOSIS ABB=ON  DNA OR ?NUCLEIC
L33       589844 SEA FILE=BIOSIS ABB=ON  GENE#
L36          276 SEA FILE=BIOSIS ABB=ON  RECEPTOR#(1A)NICOTINIC(1A)(ALPHA7 OR
                 (ALPHA 7))
L38           33 SEA FILE=BIOSIS ABB=ON  L36(8A)(L29 OR L33)
L39           12 SEA FILE=BIOSIS ABB=ON  L38 NOT PY>1996


FILE 'WPIDS' ENTERED AT 11:52:11 ON 14 OCT 1999
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FILE LAST UPDATED: 11 OCT 1999              <19991011/UP>
>>>UPDATE WEEKS:
MOST RECENT DERWENT WEEK            199941   <199941/DW>
DERWENT WEEK FOR CHEMICAL CODING:   199941
DERWENT WEEK FOR POLYMER INDEXING:  199941
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L40            4 SEA FILE=WPIDS ABB=ON  RECEPTOR#(1A)NICOTINIC(1A)(ALPHA7 OR
                 (ALPHA 7))


=> dup rem 127,139,111,140
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PROCESSING COMPLETED FOR L27
PROCESSING COMPLETED FOR L39
PROCESSING COMPLETED FOR L11
PROCESSING COMPLETED FOR L40
L41 37 DUP REM L27 L39 L11 L40 (4 DUPLICATES REMOVED)

=> d ibib ab l41 1-37; fil hom

L41 ANSWER 1 OF 37 CAPLUS COPYRIGHT 1999 ACS DUPLICATE 1
ACCESSION NUMBER: 1999:286080 CAPLUS
DOCUMENT NUMBER: 130:307552
TITLE: Structure of human gene for neuronal nicotinic receptor .alpha.7 subunit and its relation to schizophrenia
INVENTOR(S): Leonard, Sherry; Freedman, Robert
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 104 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9920757	A2	19990429	WO 1998-US21762	19981015

W: AU, CA, JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE

PRIORITY APPLN. INFO.: US 1997-956518 19971023

AB Genomic DNA sequences encoding the neuronal .alpha.7 subunit of human nicotinic acetylcholine receptor are provided, including intron-exon boundary segments and alternative spliced RACE products. The sequences of the invention may be used in the treatment and diagnosis of schizophrenia and other psychoses, and in the prepn. of transgenic animal models. Methods and compns. are provided for analyzing samples from patients suspected of suffering from diverse conditions, including epilepsy, small cell lung carcinoma and other nicotine-dependent diseases, Prader-Willi, Angelman's syndrome, and other genetic disorders, etc.

L41 ANSWER 2 OF 37 WPIDS COPYRIGHT 1999 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1999-190576 [16] WPIDS
DOC. NO. CPI: C1999-056105
TITLE: Composition for reducing side effects associated with tobacco withdrawal - comprises benzylidene- or cinnamylidene- anabaseine compounds.
DERWENT CLASS: B03
INVENTOR(S): DAY, A L; DEFIEBRE, C M; KEM, W R; MEYER, E; PAPKE, R; VAN HAAREN, F; ZOLTEWICZ, J A
PATENT ASSIGNEE(S): (UYFL) UNIV FLORIDA
COUNTRY COUNT: 19
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9910338	A2	19990304	(199916)*	EN	92

RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: JP

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9910338	A2	WO 1998-US17850	19980828

PRIORITY APPLN. INFO: US 1997-924008 19970829

AB WO 9910338 A UPAB: 19990424
NOVELTY - Composition containing benzylidene- or cinnamylidene-anabaseine targets the **alpha 7 nicotinic receptor** subtype with little or no activation of the alpha 4 beta 2 or other receptor subtypes. DETAILED DESCRIPTION - Composition comprises a benzylidene- or cinnamyl-idene-anabaseine compound of formula (I) or its salts. R1, R6, R7 = H or 1-4C alkyl; R2 = CHCH=CHX; X = a group of formula (i); R3-R5 = H, 1-4C alkyl (optionally substituted by N,N-di(1-4C)alkylamino), 1-6C alkoxy (optionally substituted by N,N-di(1-4C)alkylamino), carbo(1-4C) alkoxy, NH2, amino(1-4C)acyl, CN, N,N-di(1-4C)alkylamino, halo, OH or NO2, provided that one of R1, R3, R4, R5, R6 or R7 is not H and where R3 is not 4-N,N-di alkylamino if R1, R4, R5, R6 and R7 are all H. An INDEPENDENT CLAIM is included for a method of treating age related learning or memory impairment comprising administration of a cinnamylidene-anabaseine, provided that the cinnamylidene-anabaseine is not 3-cinnamylidene anabaseine or 3-(4-dialkylamino-cinnamylidene)anabaseine.

USE - (I) can be used for moderating or preventing tobacco-withdrawal effects; stimulating brain alpha 7 receptors antagonised by ethanol; protecting against cell loss induced by ischaemia especially cell loss from focal ischaemic insult and treating age related learning and memory impairment. The effect of alpha 7 activation by dimethoxy benzylidene anabaseine (DMXB) on NMDA triggered excitotoxicity was evaluated. Activation of **alpha 7 nicotinic receptors** was previously found to protect neurons against apoptotic degeneration caused by trophic factor deprivation. The selective alpha 7 receptor agonist DMXB also protects rat primary neocortical neurons against NMDA-receptor mediated excitotoxicity. Dose-related DMXB-induced neuroprotection was observed when administered 24 hours before, but not concomitantly, with excitatory amino acids. This was blocked by nicotinic but not muscarinic antagonist DMXB. Protection was observed primarily in penumbral areas and was blocked with the nicotinic antagonist mecamylamine, indicating that alpha 7 receptor activation is neuroprotective. MECHANISM OF ACTION - **alpha 7 Nicotinic receptor** agonist.

ADVANTAGE - (I) target the **alpha 7 nicotinic receptor** subtype with little or no activation of the alpha 4b2 or other receptor subtypes. (I) have fewer side effects than less specific receptor agonists.

Dwg.0/15

L41 ANSWER 3 OF 37 CAPLUS COPYRIGHT 1999 ACS

ACCESSION NUMBER:	1999:180477 CAPLUS
DOCUMENT NUMBER:	131:30491
TITLE:	Retrotransposons transcribed preferentially in proximal tubules of salt-hypertensive rats
AUTHOR(S):	Nagase, Miki; Kato, Akira; Ono, Toshihiro; Suzuki, Yoshiro; Hirose, Shigehisa; Fujita, Toshiro
CORPORATE SOURCE:	Fourth Department of Internal Medicine, University of Tokyo School of Medicine, Tokyo, Japan
SOURCE:	Kidney Int. (1999), 55(3), 995-1004 CODEN: KDYIA5; ISSN: 0085-2538
PUBLISHER:	Blackwell Science, Inc.

DOCUMENT TYPE: Journal
LANGUAGE: English
AB The kidney is considered to play an important etiol. role in salt-sensitive hypertension. The aim of the present study was to isolate genes whose expression differs between the kidneys of salt-hypertensive and control rats using an mRNA differential display method. Dahl salt-sensitive (DS) and control salt-resistant rats (DR) were fed a 0.3% or 8% NaCl diet. Renal RNA was amplified by RNA arbitrarily primed polymerase chain reaction (RAP-PCR) and compared among DR 0.3%, DR 8%, DS 0.3%, and DS 8%. Gene expression and localization were examd. by Northern blotting, RNase protection assay, and in situ hybridization. Full-length nucleotide sequence was detd. by screening a DS rat kidney cDNA library. The authors identified one differentially displayed clone, and its expression was greater in DS than DR, which was not affected by salt loading. The sequence was 90% homologous to the 3'-noncoding region of the nicotinic acetylcholine receptor .alpha.7 subunit gene. Its expression was kidney-specific, and was localized in the proximal tubules. The transcript level was markedly increased precedent to the development of hypertension. Its expression was also high in other salt-sensitive rats, and low in normotensive Sprague-Dawley and Wistar rats. The full-length cDNA contained elements homologous to the retroviral pol gene, a primer binding site sequence for reverse transcriptase, and long-terminal repeats. Thus, the newly identified transcripts (REPT1) belong to a novel retrotransposon family, which showed unique strain-, age-, tissue-, and cell type-specific expression pattern.

L41 ANSWER 4 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1999:167402 CAPLUS
DOCUMENT NUMBER: 131:17383
TITLE: Regional distribution of nicotinic receptor subunit mRNAs in human brain: comparison between Alzheimer and normal brain
AUTHOR(S): Hellstrom-Lindahl, Ewa; Mousavi, Malahat; Zhang, Xiao; Ravid, Ritva; Nordberg, Agneta
CORPORATE SOURCE: Karolinska Institute, Division of Molecular Neuropharmacology, Occupational Therapy and Elderly Care Research, Department of Clinical Neuroscience, Huddinge University Hospital, Huddinge, S-14186, Swed.
SOURCE: Mol. Brain Res. (1999), 66(1,2), 94-103
CODEN: MBREE4; ISSN: 0169-328X
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The regional expression of mRNA for the nicotinic acetylcholine receptor (nAChR) subunits .alpha.3, .alpha.4 and .alpha.7 was examd. in postmortem brain tissues from controls and patients with Alzheimer's disease (AD) by using quant. RT-PCR. In parallel, the nos. of nAChRs were measured by receptor binding. Relative quantification of the nAChR gene transcripts in control brains showed that expression of .alpha.3 was highest in the parietal cortex, frontal cortex and hippocampus, and lower in the temporal cortex and cerebellum. The highest level of .alpha.4 mRNA was found in the temporal cortex and cerebellum, while .alpha.7 mRNA was equally distributed in all brain regions except for hippocampus where it was less abundant. In comparison with AD brains, no differences in the expression of .alpha.3 and .alpha.4 in the temporal cortex, hippocampus and cerebellum were found. The level of .alpha.7 mRNA was significantly higher in the hippocampus of AD brains compared to controls. The binding sites for [3H] epibatidine and [3H] nicotine in the temporal cortex and [125I] .alpha.-bungarotoxin in hippocampus were significantly decreased in AD patients compared to controls. Satn. anal. of [3H] epibatidine binding revealed two classes of binding sites, with a significant redn. of the

higher affinity epibatidine binding sites in the temporal cortex of AD brain. The results show that there is a regional distribution of the expression of the different nAChRs subunits in human brain. The findings that the .alpha.3 and .alpha.4 mRNA levels were not changed in AD brains suggest that the loss of higher affinity epibatidine binding sites obsd. in AD patients cannot be attributed to alternations at the transcriptional level of the .alpha.3 and .alpha.4 genes and that causes have to be searched for at the translational and/or posttranslational level. The increased mRNA level of .alpha.7 previously found in lymphocytes, and now also in the hippocampus of AD patients, indicate that subunit specific changes in gene expression of nAChRs is assocd. with AD.

L41 ANSWER 5 OF 37 CAPLUS COPYRIGHT 1999 ACS DUPLICATE 2
ACCESSION NUMBER: 1998:479554 CAPLUS
DOCUMENT NUMBER: 129:105244
TITLE: Construction, characterization, and cloning of cDNA encoding a V274T variant human nicotinic acetylcholine receptor .alpha.-7 subunit, and methods of prodn. and use thereof
INVENTOR(S): Briggs, Clark A.; Gopalakrishnan, Murali; McKenna, David G.; Monteggia, Lisa M.; Roch, Jean-Marc; Sullivan, James P.; Touma, Edward
PATENT ASSIGNEE(S): Abbott Laboratories, USA
SOURCE: PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9828331	A2	19980702	WO 1997-US23405	19971222
WO 9828331	A3	19981105		
W: CA, JP, MX				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 946726	A2	19991006	EP 1997-954155	19971222
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1996-771737 19961220
WO 1997-US23405 19971222

AB A variant human .alpha.7 nicotinic acetylcholine receptor (nAChR) polypeptide is provided wherein the variant contains an amino acid substitution at the valine-274 position of the wild-type human .alpha.7 nAChR. **Nucleic** acid mols. encoding the variant human .alpha.7 nAChR, vectors and host cells contg. such **nucleic** acid mols. are also provided. In addn., methods are provided for producing the variant as are methods of using such variants for screening compds. for activity at the nAChR. Cloning vectors are constructed which contain sequences coding for the V274T variant. Suitable host cells are bacteria, mammalian cells, yeast, and an amphibian cell. The variant receptor protein can be used to test cytoprotective compds. for cytotoxicity, or to test for nicotinic receptor-modulating proteins. Monoclonal antibodies for the variant receptor are also claimed.

L41 ANSWER 6 OF 37 WPIDS COPYRIGHT 1999 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1998-377228 [32] WPIDS
DOC. NO. CPI: C1998-114472
TITLE: New 1,2-di substituted tetra hydro-pyridine derivatives - acting on nicotinic receptors, used for treating memory disorders associated with cerebral ageing or

neuro-degenerative disease.
DERWENT CLASS: B03
INVENTOR(S): COMPERE, D; DAS, B C; LEPAGNOL, J; MARAZANO, C; DAS, C B
PATENT ASSIGNEE(S): (ADIR) ADIR & CIE
COUNTRY COUNT: 29
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9824765	A1	19980611	(199832)*	FR	34
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: AU BR CA CN HU JP NO NZ PL US					
FR 2756826	A1	19980612	(199832)		
ZA 9710960	A	19980826	(199840)		29
AU 9876243	A	19980629	(199845)		
NO 9902731	A	19990604	(199937)		
EP 937043	A1	19990825	(199939)	FR	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9824765	A1	WO 1997-FR2172	19971202
FR 2756826	A1	FR 1996-14951	19961205
ZA 9710960	A	ZA 1997-10960	19971205
AU 9876243	A	AU 1998-76243	19971202
NO 9902731	A	WO 1997-FR2172	19971202
		NO 1999-2731	19990604
EP 937043	A1	EP 1997-948974	19971202
		WO 1997-FR2172	19971202

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9876243	A	Based on	WO 9824765
EP 937043	A1	Based on	WO 9824765

PRIORITY APPLN. INFO: FR 1996-14951 19961205
AB WO 9824765 A UPAB: 19981021
Tetrahydropyridine derivatives of formula (I) and their isomers and salts with acids and bases are new. X = H and Y = OH; or X + Y = O; R = alkyl or phenyl (optionally substituted by one or more of halo, alkyl, alkoxy, polyhaloalkyl and OH); R1 = H or -CH2-C(X')(Y')-R3; X' = H and Y' = OH; or X' + Y' = O or 1-4C alkylenedioxy; or X,Y = alkoxy; R3 = alkoxy or phenyl (optionally substituted by one or more of halo, alkyl, alkoxy, polyhaloalkyl and OH); R2 = H or alkyl (optionally substituted by one or more of aryl (optionally substituted), alkoxy and OH); provided that if R = R2 = Me, X = H, Y = OH and R1 = -CH2CH(OH)R3, then R3 is not phenyl; alkyl moieties have 1-6C.

USE - (I) interact specifically with nicotinic receptors, especially having preferential affinity for **alpha 7 nicotinic receptors**. They are used for the treatment of memory disorders associated with cerebral ageing and neurodegenerative diseases (such as Alzheimer's, Parkinson's, Pick's and Korsakoff's diseases, and frontal and sub-cortical dementia) (all claimed). Dwg.0/0

L41 ANSWER 7 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1998:541320 CAPLUS

DOCUMENT NUMBER: 129:240696
TITLE: GC- and E-box motifs as regulatory elements in the proximal promoter region of the neuronal nicotinic receptor .alpha.7 subunit gene
AUTHOR(S): Carrasco-Serrano, Carmen; Campos-Caro, Antonio; Viniegra, Salvador; Ballesta, Juan J.; Criado, Manuel
CORPORATE SOURCE: Department of Neurochemistry, Universidad Miguel Hernandez, Alicante, 03550, Spain
SOURCE: J. Biol. Chem. (1998), 273(32), 20021-20028
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The .alpha.7 subunit is a component of .alpha.-bungarotoxin-sensitive nicotinic acetylcholine receptors expressed in bovine adrenomedullary chromaffin cells. The proximal promoter of the gene coding for this subunit contains several GC-boxes and one E-box. Deletion anal. and transient transfections showed that a 120-base pair region (-77 to +43) including all of these elements gave rise to .apprx.70 and 95% of the maximal transcriptional activity obsd. in chromaffin and SHSY-5Y neuroblastoma cells, resp. Site-directed mutagenesis of the different elements indicated that both GC and E motifs contribute to the activity of the .alpha.7 gene in a very prominent way. Using electrophoretic mobility shift assays, the upstream stimulatory factor (USF) was shown to be a component of the complexes that interacted with the E-box when nuclear exts. from chromaffin and SHSY-5Y cells were used. Binding of the early growth response gene transcription factor (Egr-1) to three different GC-boxes was also demonstrated by shift assays and DNase I footprint anal. Likewise, .alpha.7 promoter activity increased by up to 5-fold when .alpha.7 constructs and an Egr-1 expression vector were cotransfected into chromaffin cell cultures. Mutagenesis of individual GC-boxes had little effect on Egr-1 activation. By contrast, pairwise suppression of GC-boxes abolished activation, esp. when the most promoter-proximal of the Egr-1 sites was removed. Taken together, these studies indicate that the .alpha.7 gene is likely to be a target for multiple signaling pathways, in which various regulatory elements are involved.

L41 ANSWER 8 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1998:303559 CAPLUS
DOCUMENT NUMBER: 129:1630
TITLE: Exposure to prenatal nicotine transiently increases neuronal nicotinic receptor subunit .alpha.7, .alpha.4 and .beta.2 messenger RNAS in the postnatal rat brain
AUTHOR(S): Shacka, J. J.; Robinson, S. E.
CORPORATE SOURCE: Department of Pharmacology and Toxicology, Medical College of Virginia, Virginia Commonwealth University, Richmond, VA, 23298-0613, USA
SOURCE: Neuroscience (Oxford) (1998), 84(4), 1151-1161
CODEN: NRSCDN; ISSN: 0306-4522
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB This study detd. the effects of prenatal nicotine exposure (2 mg/kg/day) in Sprague-Dawley CD rats via s.c. implanted osmotic mini pumps, during gestational days 7-21, on postnatal levels of neuronal nicotinic receptor .alpha.4, .alpha.7 and .beta.2 subunit mRNAs. Northern anal. of postnatal day 1, 7, 14 and 28 hippocampal/septal and cortical total RNA using .alpha.-[32P]dCTP-labeled .alpha.4, .alpha.7 and .beta.2 complementary DNA probes identified a single (5.7-kb) .alpha.7 mRNA, three (2.4-, 3.8- and 8.0-kb) .alpha.4 mRNAs and four (3.7-, 5.0-, 7.5- and 10.0-kb) .beta.2

mRNAs. In comparison to prenatal saline, prenatal nicotine produced several significantly higher mRNA levels (cortical: 5.7-kb .alpha.7, 2.4-, 3.8- and 8.0-kb .alpha.4, 10.0-kb .beta.2; hippocampal/septal: 2.4- and 8.0-kb .alpha.4); these increases occurred predominantly on, but were not restricted to, postnatal day 14. Effects of nicotine were generally resolved by postnatal day 28. Collapsing the data across sex and age, a significant treatment effect indicated that hippocampal/septal and cortical 8.0-kb .alpha.4 mRNA levels and 10.0-kb .beta.2 mRNA levels were significantly higher following prenatal nicotine exposure. This is the first study indicating that prenatal nicotine produces alterations in developing postnatal rat neuronal nicotinic receptor mRNA levels, possibly by premature stimulation of neuronal nicotinic receptors. These results further implicate the teratogenic potential of nicotine in postnatal neuronal development.

L41 ANSWER 9 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1998:168315 CAPLUS
DOCUMENT NUMBER: 128:267148
TITLE: Sensitivity to the seizure-inducing effects of nicotine is associated with strain-specific variants of the .alpha.5 and .alpha.7 nicotinic receptor subunit genes
AUTHOR(S): Stitzel, Jerry A.; Blanchette, Jennifer M.; Collins, Allan C.
CORPORATE SOURCE: Institute for Behavioral Genetics, University of Colorado, Boulder, CO, USA
SOURCE: J. Pharmacol. Exp. Ther. (1998), 284(3), 1104-1111
CODEN: JPETAB; ISSN: 0022-3565
PUBLISHER: Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Restriction fragment length polymorphisms (rflps) have been identified for the nicotinic ACh receptor subunit genes .alpha.5 and .alpha.7 between two mouse strains (C3H/2ibg and DBA/2ibg) that differ in sensitivity to the convulsant effects of nicotine. In the study reported here, F2 animals derived from these two parental stains were tested for their sensitivity to the convulsant effects of nicotine as measured by seizure frequency and over-all sensitivity score. Subsequently, the animals were genotyped for the .alpha.5 and .alpha.7 rflps. In addn., levels of .alpha.-bungarotoxin (.alpha.-BTX) binding were measured in four brain regions (colliculi, hippocampus, hypothalamus and striatum) to det. whether there is a correlation among .alpha.-BTX binding levels, sensitivity to nicotine and nicotinic ACh receptor subunit genotype. A significant relationship was obsd. between .alpha.5 and .alpha.7 genotype and sensitivity to nicotine. In addn., the .alpha.7 rflp significantly correlated with levels of .alpha.-BTX binding in hippocampus, colliculi and striatum. The .alpha.5 rflp did not correlate with .alpha.-BTX binding levels in any brain region. Levels of .alpha.-BTX binding did not correlate with nicotine-induced seizure sensitivity or overall nicotine sensitivity score in any of the four brain regions examd.

L41 ANSWER 10 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1999:33942 CAPLUS
DOCUMENT NUMBER: 130:218613
TITLE: Postnatal changes of nicotinic acetylcholine receptor .alpha.2, .alpha.3, .alpha.4, .alpha.7 and .beta.2 subunits genes expression in rat brain
AUTHOR(S): Zhang, Xiao; Liu, Chuan; Miao, Hui; Gong, Ze-Hui; Nordberg, Agneta
CORPORATE SOURCE: Department of Clinical Neuroscience and Family Medicine, Huddinge University Hospital, Huddinge, 141

86, Swed.
SOURCE: Int. J. Dev. Neurosci. (1998), 16(6), 507-518
CODEN: IJDND6; ISSN: 0736-5748
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Postnatal changes of nicotinic acetylcholine receptor (nAChR) .alpha.2, .alpha.3, .alpha.4, .alpha.7 and .beta.2 subunits mRNAs were investigated in rat brain using RNase protection assay. Multiple developmental patterns were obsd.: transient expression during the first few postnatal weeks; .alpha.2 in the hippocampus and brain stem, .alpha.3 in the striatum, cerebellum and cortex, .alpha.4 in the hippocampus, striatum and cerebellum, .alpha.7 in the cerebellum and .beta.2 in the striatum. Const. expression across development; .alpha.2 and .alpha.3 in the thalamus, .alpha.4 in the cortex, thalamus and brain stem, .alpha.7 in the thalamus and brain stem and .beta.2 in all brain regions except striatum. Non-detection across development; .alpha.2 in the cortex, striatum and cerebellum. Increase with age; .alpha.7 in the cortex and hippocampus. Bell-shaped development; .alpha.7 in the striatum. Postnatal changes of nAChR isoforms in different brain regions of rat were investigated by receptor binding assays. The developmental patterns of [3H]epibatidine and (-)-[3H]nicotine binding sites were similar to each other in each brain region, but different from that of [3H].alpha.-bungarotoxin binding sites. No obvious correlation was obsd. between the developmental patterns of [3H].alpha.-bungarotoxin, [3H]epibatidine and (-)-[3H]nicotine binding sites and corresponding subunits mRNAs. These results indicate that multiple mechanisms are involved in changes of gene expression of nAChRs subunits in the brain of developing rats.

L41 ANSWER 11 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1998:649507 CAPLUS
DOCUMENT NUMBER: 130:48196
TITLE: On the transcriptional regulation of neuronal nA ChR genes
AUTHOR(S): Matter, Jean-Marc; Matter-Sadzinski, Lidia; Roztocil, Tomas; Hernandez, Marie-Clemencia; Couturier, Sabine; Ong, Ming-Thong; Ballivet, Marc
CORPORATE SOURCE: Department of Biochemistry, University of Geneva, Geneva, CH-1211/4, Switz.
SOURCE: J. Physiol. (Paris) (1998), 92(3-4), 245-248
CODEN: JHYSEM; ISSN: 0928-4257
PUBLISHER: Editions Scientifiques et Medicales Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The promoters driving transcription of the neuronal nicotinic genes .alpha.7 and .beta.3 have been characterized in the chicken. Although their regulatory modalities are thoroughly different, they nevertheless lead to co-expression in the same neurons.

L41 ANSWER 12 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1998:694126 CAPLUS
DOCUMENT NUMBER: 130:61849
TITLE: Genomic organization and partial duplication of the human .alpha.7 neuronal nicotinic acetylcholine receptor gene (CHRNA7)
AUTHOR(S): Gault, Judith; Robinson, Misi; Berger, Ralph; Drebing, Carla; Logel, Judith; Hopkins, Jan; Moore, Ted; Jacobs, Suzette; Meriwether, Jennifer; Choi, Mun Jun; Kim, Eun Jung; Walton, Katy; Buiting, Karin; Davis, Ashley; Breese, Charles; Freedman, Robert; Leonard, Sherry

CORPORATE SOURCE: Dep. of Psychiatry and Dep. of Pharmacology, University of Colorado Health Sciences Center, Denver, CO, 80262, USA
SOURCE: Genomics (1998), 52(2), 173-185
CODEN: GNMCEP; ISSN: 0888-7543
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The human .alpha.7 neuronal nicotinic acetylcholine receptor gene (HGMW-approved symbol CHRNA7) has been characterized from genomic clones. The gene is similar in structure to the chick .alpha.7 gene with 10 exons and conserved splice junction positions. The size of the human gene is estd. to be larger than 75 kb. A putative promoter 5' of the translation start in exon 1 has been cloned and sequenced. The promoter region lacks a TAT box and has a high GC content (77%). Consensus Sp1, Ap-2, Egr-1, and CREB transcription factor binding sites appear to be conserved between bovine and human genes. The .alpha.7 nAChR gene was found to be partially duplicated, with both loci mapping to the chromosome 15q13 region. A yeast artificial chromosome contig was constructed over a genetic distance of 5 cM that includes both .alpha.7 loci and the region between them. Four novel exons are described, located in genomic clones contg. the partially duplicated gene. The duplicated sequences, including the novel exons, are expressed in human brain. (c) 1998 Academic Press.

L41 ANSWER 13 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1998:415559 CAPLUS
DOCUMENT NUMBER: 129:157208
TITLE: Postnatal developmental regulation of neuronal nicotinic receptor subunit .alpha.7 and multiple .alpha.4 and .beta.2 mRNA species in the rat
AUTHOR(S): Shacka, John J.; Robinson, Susan E.
CORPORATE SOURCE: Box 980613, Medical College of Virginia, Department of Pharmacology and Toxicology, Virginia Commonwealth University, Richmond, VA, 23298-0613, USA
SOURCE: Dev. Brain Res. (1998), 109(1), 67-75
CODEN: DBRRDB; ISSN: 0165-3806
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB This study examd. the postnatal development of neuronal nicotinic receptor (nAChR) .alpha.7, .alpha.4 and .beta.2 subunit mRNA in the Sprague Dawley rat brain. The hippocampus, septum and cortex were removed on postnatal day 1 (P1), P7, P14, or P28 and analyzed by sex. Northern anal. of cortical and pooled hippocampal and septal total RNA with 32P-.alpha.-dCTP-labeled .alpha.7, .alpha.4 (recognizing .alpha.4.1 and .alpha.4.2 mRNA), and .beta.2 nAChR cDNA probes identified three (2.4, 3.8 and 8.0 kb) .alpha.4, four (3.5, 5.0, 7.5 and 10.0 kb) .beta.2 and a single 5.7 kb .alpha.7 mRNA species. Cortical .alpha.4 mRNA peaked on P14 and remained high on P28, whereas hippocampal/septal .alpha.4 mRNA was higher on P7 and P14 than on P1 and P28. Expression of cortical and hippocampal/septal .beta.2 mRNAs decreased on P7, followed by a dramatic peak on P14. .alpha.7 MRNA peaked on P7. Throughout development, 2.4 kb .alpha.4 mRNA was more intense than 3.8 kb .alpha.4 mRNA, whereas 5.0 kb .beta.2 mRNA was the most intense cortical and hippocampal/septal .beta.2 mRNA species. The .alpha.4.1-specific cDNA probe detected similar-sized .alpha.4 bands as the pan-specific .alpha.4 cDNA probe, therefore precluding the identification of any band as .alpha.4.2-specific. These results suggest that postnatal expression of .alpha.4 and .alpha.7 but not .beta.2 mRNAs is brain region-specific, and that the contribution of multiple nAChR subunit mRNA species in development may vary.

L41 ANSWER 14 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1998:152373 CAPLUS
DOCUMENT NUMBER: 128:266840
TITLE: Genetic analysis and characterization of the rat neuronal nicotinic acetylcholine receptor (alpha)7 subunit gene
AUTHOR(S): Nagavarapu, Usha
CORPORATE SOURCE: Ohio State Univ., Columbus, OH, USA
SOURCE: (1997) 131 pp. Avail.: UMI, Order No. DA9813322 From: Diss. Abstr. Int., B 1998, 58(10), 5282
DOCUMENT TYPE: Dissertation
LANGUAGE: English
AB Unavailable

L41 ANSWER 15 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1997:736608 CAPLUS
DOCUMENT NUMBER: 128:33062
TITLE: Nicotinic acetylcholine receptors of muscle and neuronal (.alpha.7) types coexpressed in a small cell lung carcinoma
AUTHOR(S): Sciamanna, Michele A.; Griesmann, Guy E.; Williams, Carol L.; Lennon, Vanda A.
CORPORATE SOURCE: Departments of Immunology, Neurology, and Laboratory Medicine and Pathology, Mayo Clinic, Rochester, MN, USA
SOURCE: J. Neurochem. (1997), 69(6), 2302-2311 CODEN: JONRA9; ISSN: 0022-3042
PUBLISHER: Lippincott-Raven Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English
AB SCC-37 is a small cell lung carcinoma line that aberrantly expresses muscle-type nicotinic acetylcholine receptors (nAChRs). It was established from a patient with a paraneoplastic autoimmune neuromuscular disorder, myasthenia gravis. When grown as a xenograft tumor, SCC-37 cells express plasma membrane receptors that bind 125I-labeled .alpha.-bungarotoxin (125I-.alpha.-BTx), cosediment with 9S nAChR pentamers, and bind to a monoclonal antibody (MAb 35) specific for muscle-type (.alpha.1 subunit) .alpha.-BTx receptors. The agonist carbamylcholine (carbachol) stimulates influx of 22Na+ in SCC-37 cells; this is inhibited by .alpha.-BTx and by d-tubocurarine. Long-term cultured SCC-37 cells have functional and ligand-binding evidence for surface coexpression of both .alpha.1 and neuronal-type (.alpha.7 subunit) .alpha.-BTx receptors. A subclone of SCC-37, designated SCC-A9, expresses only the neuronal-type (.alpha.7 subunit) .alpha.-BTx receptors on its surface. Carbachol does not stimulate 22Na+ influx in SCC-A9 cells, but cytisine initiates a sustained influx of Ca2+. Activation of this response is inhibited by .alpha.-BTx and by the .alpha.7-selective antagonist methyllycaconitine. Addn. of Co2+ abrogates the sustained elevation of intracellular free Ca2+ concn., implying that the cytisine-stimulated influx of Ca2+ is sustained by secondary opening of voltage-sensitive channels in the plasma membrane. Surface receptors for 125I-.alpha.-BTx are blocked by methyllycaconitine and d-tubocurarine. Solubilized .alpha.-BTx receptors from plasma membranes of SCC-A9 cells cosediment with 10S neuronal nAChR pentamers and bind to an .alpha.7-specific monoclonal antibody (MAb P27) but not to the muscle nAChR-reactive MAb 35. However, MAb P27 and MAb 35 both bind to .alpha.-BTx receptors solubilized from the cytoplasmic compartments of SCC-A9 and the parental SCC-37 line. Reverse transcription-PCR anal. revealed RNA transcripts for .alpha.7 and .alpha.1 subunits in both SCC-A9 and SCC-37 cells. The nAChRs that are expressed in these novel human cell lines can regulate cation fluxes directly as well as indirectly by

synergizing with the activity of voltage-sensitive Ca2+ channels. These activities may influence the secretion of autocrine growth factors and the transcription of growth regulatory genes and thus be pertinent to the growth and metastasis of malignant neuroendocrine neoplasms.

L41 ANSWER 16 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1997:82865 CAPLUS
DOCUMENT NUMBER: 126:166742
TITLE: Expression of the nicotinic receptor .alpha.7 gene in tendon and periosteum during early development
AUTHOR(S): Romano, Suzanne J.; Corriveau, Roderick A.; Schwarz, Richard I.; Berg, Darwin K.
CORPORATE SOURCE: Department of Biology, University of California-San Diego, La Jolla, CA, USA
SOURCE: J. Neurochem. (1997), 68(2), 640-648
CODEN: JONRA9; ISSN: 0022-3042
PUBLISHER: Lippincott-Raven
DOCUMENT TYPE: Journal
LANGUAGE: English
AB One of the most abundant nicotinic acetylcholine receptors expressed in the central and peripheral nervous systems is a species that contains the .alpha.7 gene product, binds .alpha.-bungarotoxin with high affinity, and has a high relative permeability to calcium. The .alpha.7 gene is also expressed at low levels in embryonic muscle tissue. We show here that the .alpha.7 gene is expressed in tendon fibroblasts and periosteal cells during development. In situ hybridizations identify .alpha.7 transcripts in tissue sections contg. embryonic tendon and periosteum. RNase protection expts. demonstrate .alpha.7 mRNA in primary tendon cells grown in culture. Immunofluorescence with subunit-specific monoclonal antibodies reveals .alpha.7 protein in embryonic tendon. Immunopptn. assays with the antibodies indicate that the .alpha.7-contg. species in tendon is capable of binding .alpha.-bungarotoxin and that a similar species can be identified at low levels on the surface of fibroblasts in culture. The results show that the .alpha.7 gene product is expressed in a range of tissues, including cells thought to be nonexcitable. The distribution of .alpha.7 expression early in development and the ability of .alpha.7-contg. receptors to elevate intracellular calcium suggest that the gene may influence a variety of calcium-dependent events during embryogenesis.

L41 ANSWER 17 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1997:77576 CAPLUS
DOCUMENT NUMBER: 126:153474
TITLE: Linkage of a neurophysiological deficit in schizophrenia to a chromosome 15 locus
AUTHOR(S): Freedman, Robert; Coon, Hilary; Myles-Worsley, Marina; Orr-Urtreger, Avi; Olincy, Ann; Davis, Ashley; Polymeropoulos, Mihael; Holik, John; Hopkins, Jan; Hoff, Mark; Rosenthal, Judy; Waldo, Merilyne C.; Reimherr, Fred; Wender, Paul; Yaw, Jeffrey; Young, David A.; Breese, Charles R.; Adams, Catherine; Patterson, David; Adler, Lawrence E.; Kruglyak, Leonid; Leonard, Sherry; Byerley, William
CORPORATE SOURCE: Department Psychiatry Pharmacology Biochemistry, University Colorado School Medicine, Denver, CO, 80262, USA
SOURCE: Proc. Natl. Acad. Sci. U. S. A. (1997), 94(2), 587-592
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Inheritance of a defect in a neuronal mechanism that regulates response to auditory stimuli was studied in nine families with multiple cases of schizophrenia. The defect, a decrease in the normal inhibition of the P50 auditory-evoked response to the second of paired stimuli, is assocd. with attentional disturbances in schizophrenia. Decreased P50 inhibition occurs not only in most schizophrenics, but also in many of their nonschizophrenic relatives, in a distribution consistent with inherited vulnerability for the illness. Neurobiol. investigations in both humans and animal models indicated that decreased function of the .alpha.7-nicotinic cholinergic receptor could underlie the physiol. defect. In the present study, a genome-wide linkage anal., assuming autosomal dominant transmission, showed that the defect is linked [max. logarithm of the odds (lod) score = 5.3 with zero recombination] to a dinucleotide polymorphism at chromosome 15q13-14, the site of the .alpha.7-nicotinic receptor. Despite many schizophrenics' extremely heavy nicotine use, nicotinic receptors were not previously thought to be involved in schizophrenia. The linkage data thus provide unique new evidence that the .alpha.7-nicotinic receptor gene may be responsible for the inheritance of a pathophysiol. aspect of the illness.

L41 ANSWER 18 OF 37 MEDLINE
ACCESSION NUMBER: 97149417 MEDLINE
DOCUMENT NUMBER: 97149417
TITLE: Activation of the recombinant human **alpha 7** nicotinic acetylcholine receptor significantly raises intracellular free calcium.
AUTHOR: Delbono O; Gopalakrishnan M; Renganathan M; Monteggia L M; Messi M L; Sullivan J P
CORPORATE SOURCE: Department of Physiology and Pharmacology, Bowman Gray School of Medicine, Wake Forest University, Winston-Salem, North Carolina, USA.
CONTRACT NUMBER: 2-P60AG10484 (NIA)
T-32-AG00182 (NIA)
K01 AG00692 (NIA)
SOURCE: JOURNAL OF PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS, (1997 Jan) 280 (1) 428-38.
Journal code: JP3. ISSN: 0022-3565.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199704
ENTRY WEEK: 19970403

AB The **alpha 7** nicotinic acetylcholine receptor (nAChR) subtype, unlike other neuronal nicotinic receptors, exhibits a relatively high permeability to Ca++ ions. Although Ca++ entry through this receptor subtype has been implicated in various Ca(++)-dependent processes in the central nervous system, little is known about how this receptor modulates mammalian intracellular Ca++ dynamics. Intracellular Ca++ responses evoked by activation of the human **alpha 7** nAChRs stably expressed in HEK-293 (human embryonic kidney) cells were studied. Inward current and intracellular Ca++ transients were recorded simultaneously in response to a fast drug application system. Current recordings under whole-cell voltage-clamp and fast ratiometric intracellular Ca++ imaging acquisition were synchronized to drug pulses. The mean peak [Ca++]i observed with 100 microM (-)-nicotine was 356 +/- 48 nM (n = 8). The magnitude of the intracellular Ca++ elevation corresponds to a 20% fractional current carried by Ca++ ions. The EC50 of the intracellular Ca++ responses for (-)-nicotine, (+/-)-epibatidine, 1,1 dimethyl-4-phenyl-piperazinium and acetylcholine were 51, 3.5, 75 and 108 microM, respectively. These EC50 values strongly correlate with those

recorded for the cationic inward current through **alpha 7** nAChR. alpha-Bungarotoxin, methyllcaconitine or extracellular Ca++ chelation ablated (-)-nicotine-evoked increase in intracellular Ca++ concentration. This study provides evidence that cation influx through the human **alpha 7** nAChR is sufficient to mediate a significant, transient, rise in intracellular Ca++ concentration.

L41 ANSWER 19 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1997:716663 CAPLUS
DOCUMENT NUMBER: 128:18929
TITLE: Comparison of the regional expression of nicotinic acetylcholine receptor .alpha.7 mRNA and [125I]-.alpha.-bungarotoxin binding in human postmortem brain
AUTHOR(S): Breese, Charles R.; Adams, Catherine; Logel, Judy; Drebing, Carla; Rollins, Yvonne; Barnhart, Michelle; Sullivan, Bernadette; Demasters, Bette K.; Freedman, Robert; Leonard, Sherry
CORPORATE SOURCE: Department of Pharmacology, University of Colorado Health Sciences Center, Denver, CO, 80262, USA
SOURCE: J. Comp. Neurol. (1997), 387(3), 385-398
CODEN: JCNEAM; ISSN: 0021-9967
PUBLISHER: Wiley-Liss
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Neuronal nicotinic acetylcholine receptors are expressed in the human central nervous system. A specific subtype of this receptor family, the .alpha.7 nicotinic acetylcholine receptor, is thought to be the principal .alpha.-bungarotoxin (.alpha.BTX)-binding protein in mammalian brain. Although the expression of this receptor subtype has been characterized in rat, no study has specifically compared the expression of both the .alpha.7 gene and the localization of BTX binding sites in human brain. Expression of .alpha.7 mRNA and receptor protein in human postmortem brain tissue was examd. by in situ hybridization and [125I]-.alpha.-bungarotoxin autoradiog., resp., with particular emphasis on regions assocd. with sensory processing. Regions with high levels of both .alpha.7 gene expression and [125I]-.alpha.BTX binding include the nucleus reticularis of the thalamus, the lateral and medial geniculate bodies, the basilar pontine nucleus, the horizontal limb of the diagonal band of Broca, the nucleus basalis of Meynert, and the inferior olivary nucleus. High-to-moderate levels of .alpha.7 probe hybridization were also seen in the hippocampus and the cerebral cortex; however, there was a reduced or variable degree of [125I]-.alpha.BTX binding in these regions compared with the level of probe hybridization. In most brain regions, [125I]-.alpha.BTX binding was localized to neuronal cell bodies similar in morphol. to those that exhibited .alpha.7 hybridization, suggesting that the high-affinity [125I]-.alpha.BTX binding sites in the human brain are likely to be principally composed of .alpha.7 receptor subtypes.

L41 ANSWER 20 OF 37 CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1997:79343 CAPLUS
DOCUMENT NUMBER: 126:129453
TITLE: Neuronal-type acetylcholine receptors and regulation of .alpha.7 gene expression in vertebrate skeletal muscle
AUTHOR(S): Romano, Suzanne J.; Pugh, Phyllis C.; Mcintosh, J. Michael; Berg, Darwin K.
CORPORATE SOURCE: Department of Biology, University of California, La Jolla, CA, 92093-0357, USA
SOURCE: J. Neurobiol. (1997), 32(1), 69-80
CODEN: JNEUBZ; ISSN: 0022-3034

PUBLISHER: Wiley
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Several neuronal nicotinic acetylcholine receptor (AChR) genes are expressed in chick skeletal muscle during development. One of the most abundantly expressed is .alpha.7, which produces a protein capable of binding .alpha.-bungarotoxin and is phys. distinct from muscle AChRs contg. the .alpha.1 gene product. The authors show here that the .alpha.7-contg. species in muscle is indistinguishable pharmacol. from .alpha.7-contg. AChRs in neurons. In addn., immunol. anal. with subunit-specific muscle antibodies shows that the .alpha.7-contg. species in muscle lacks the .beta.1 and .delta. muscle AChR gene products as it does the .alpha.1. RNase protection expts. measuring .alpha.7 mRNA levels indicate that the .alpha.1 and .alpha.7 genes may, in part, be subject to similar kinds of regulation in the tissue. Surgical denervation of leg muscle in newly hatched chicks caused a small and transient increase in .alpha.7 mRNA after 8 days, while .alpha.1 transcripts underwent a large and sustained increase in no. Similarly, treating myotube cultures with tetrodotoxin caused a modest increase in .alpha.7 transcript levels and a large increase in .alpha.1. Calcitonin gene-related peptide (CGRP) increased both kinds of transcripts in myotube cultures equally as did treatment with 8-bromo-cAMP; CGRP is thought to work via a cAMP-dependent pathway in muscle. In at least one respect, however, .alpha.7 expression in muscle differs qual. from that of .alpha.1: AChR-inducing activity (ARIA) increased .alpha.1 mRNA levels in culture while slightly depressing .alpha.7 mRNA levels. The regulatory pattern of .alpha.7 expression in muscle may combine features of both .alpha.7 expression in neurons and .alpha.1 expression in muscle.

L41 ANSWER 21 OF 37 BIOSIS COPYRIGHT 1999 BIOSIS
ACCESSION NUMBER: 1996:551830 BIOSIS
DOCUMENT NUMBER: PREV199699274186
TITLE: Characterization of the rat neuronal **nicotinic** acetylcholine **receptor alpha-7** subunit **gene**.
AUTHOR(S): Nagavarapu, U.; Boyd, R. T.
CORPORATE SOURCE: Dep. Pharmacol., Ohio State Univ. Coll. Med., Columbus, OH 43210 USA
SOURCE: Society for Neuroscience Abstracts, (1996) Vol. 22, No. 1-3, pp. 1261.
Meeting Info.: 26th Annual Meeting of the Society for Neuroscience Washington, D.C., USA November 16-21, 1996
ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English

L41 ANSWER 22 OF 37 BIOSIS COPYRIGHT 1999 BIOSIS
ACCESSION NUMBER: 1996:551827 BIOSIS
DOCUMENT NUMBER: PREV199699274183
TITLE: Generation of mice deficient in the **alpha7** neuronal **nicotinic receptor gene** by targeted recombination.
AUTHOR(S): Orr-Urtreger, Avi (1); Goldner, Finn; Patrick, Jim; Beaudet, Art
CORPORATE SOURCE: (1) Dep. Molecular Human Genetics, The Howard Hughes Med. Inst., Houston, TX 77030 USA
SOURCE: Society for Neuroscience Abstracts, (1996) Vol. 22, No. 1-3, pp. 1260.
Meeting Info.: 26th Annual Meeting of the Society for Neuroscience Washington, D.C., USA November 16-21, 1996
ISSN: 0190-5295.

DOCUMENT TYPE: Conference
LANGUAGE: English

L41 ANSWER 23 OF 37 BIOSIS COPYRIGHT 1999 BIOSIS
ACCESSION NUMBER: 1996:476469 BIOSIS
DOCUMENT NUMBER: PREV199699206025
TITLE: Nicotinic receptor function in schizophrenia.
AUTHOR(S): Leonard, Sherry (1); Adams, Catherine; Breese, Charles R.; Adler, Lawrence E.; Bickford, Paula; Byerley, William; Coon, Hilary; Griffith, Jay M.; Miller, Christine; Myles-Worsley, Marina; Nagamoto, Herbert T.; Rollins, Yvonne; Stevens, Karen E.; Waldo, Merilyn; Freedman, Robert
CORPORATE SOURCE: (1) Dep. Pharmacol., Univ. Colorado Health Sci. Cent., Box C-268-71, 4200 E. 9th Ave., Denver, CO 80262 USA
SOURCE: Schizophrenia Bulletin, (1996) Vol. 22, No. 3, pp. 431-445. ISSN: 0586-7614.
DOCUMENT TYPE: General Review
LANGUAGE: English
AB Schizophrenia can be partially characterized by deficits in sensory processing. Biochemical, molecular, and genetic studies of one such endophenotype, the P50 auditory-evoked potential gating deficit, suggest that one of the neuronal nicotinic receptors, the alpha-7 nicotinic receptor, may function in an inhibitory neuronal pathway involved in this phenotype. The P50 deficit is normalized in nongating subjects by nicotine. Although most schizophrenia patients are heavy smokers, the effects of nicotine may be transient, as alpha-7 receptors are known to desensitize rapidly. In an animal model of the P50 gating deficit, antagonists of the alpha-7 nicotinic receptor block normal gating of the second of paired auditory stimuli. Regional localization of receptor expression includes areas known to function in sensory filtering. An inhibitory mechanism, in the hippocampus, may involve nicotinic stimulation of gamma-aminobutyric acid (GABA)ergic interneurons, resulting in decreased response to repetitive stimuli. Expression of the alpha-7 receptor is decreased in hippocampal brain tissue, dissected postmortem, from schizophrenia subjects. The P50 deficit is inherited in schizophrenia pedigrees, but it is not sufficient for disease development and thus represents a predisposition factor. Linkage analysis between the P50 deficit in multiplex schizophrenia pedigrees and deoxyribonucleic acid (DNA) markers throughout the genome yielded positive lod scores to DNA markers mapping to a region of chromosome 15 containing the **alpha -7 nicotinic receptor gene.**
Elucidation of possible interactions of the P50 with other factors, known to be important in the etiology of the disease, is important in determining an overall pathobiology of schizophrenia.

L41 ANSWER 24 OF 37 MEDLINE
ACCESSION NUMBER: 97062879 MEDLINE
DOCUMENT NUMBER: 97062879
TITLE: Comparative structure of human neuronal alpha 2-**alpha 7** and beta 2-beta 4 nicotinic acetylcholine receptor subunits and functional expression of the alpha 2, alpha 3, alpha 4, **alpha 7** , beta 2, and beta 4 subunits.
AUTHOR: Elliott K J; Ellis S B; Berckhan K J; Urrutia A; Chavez-Noriega L E; Johnson E C; Velicelebi G; Harpold M M
CORPORATE SOURCE: SIBIA Neurosciences Inc., La Jolla, CA, USA.
SOURCE: JOURNAL OF MOLECULAR NEUROSCIENCE, (1996 Fall) 7 (3) 217-28.
Journal code: AVM. ISSN: 0895-8696.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U62431; GENBANK-U62432; GENBANK-U62433; GENBANK-U62434; GENBANK-U62435; GENBANK-U62436; GENBANK-U62437; GENBANK-U62438; GENBANK-U62439
ENTRY MONTH: 199707
AB cDNA clones encoding human neuronal nicotinic acetylcholine receptor alpha 2, alpha 3, alpha 4, alpha 5, alpha 6, **alpha 7**, beta 2, beta 3, and beta 4 subunits were isolated from brainstem, hippocampus, prefrontal cortex, substantia nigra, thalamus, and IMR32 libraries. Human alpha 2 and alpha 6 and full-length beta 3 and beta 4 clones have not been previously reported. Deduced amino acid sequences of the alpha 2, alpha 6, beta 3, and beta 4 predicted mature peptides are 503 residues (56.9 kDa), 464 residues (53.7 kDa), 440 residues (50.8 kDa), and 477 residues (54.1 kDa), respectively. These sequences show 84 (alpha 2), 87 (alpha 6), 89 (beta 3), and 84% (beta 4) identity to the corresponding rat sequences. The amino termini of the human alpha 2 and beta 3 mature peptides contain 23 and six additional residues, respectively, compared to those of rat alpha 2 and beta 3. Recombinant receptors were expressed in Xenopus laevis oocytes injected with in vitro transcripts encoding either **alpha 7** alone or alpha 2, alpha 3, or alpha 4 in pairwise combination with beta 2 or beta 4. Inward currents were elicited by the application of acetylcholine (1-100 microM) and other agonists; these responses were blocked 65-97% by application of 10 microM d-tubocurare, confirming functional expression of human nicotinic receptors.

L41 ANSWER 25 OF 37 BIOSIS COPYRIGHT 1999 BIOSIS
ACCESSION NUMBER: 1996:555255 BIOSIS
DOCUMENT NUMBER: PREV199699277611
TITLE: A novel hypersynchronous neocortical EEG phenotype in mice deficient in the neuronal **nicotinic** acetylcholine **receptor** (nAChRs) **alpha-7** subunit **gene**.
AUTHOR(S): Orr-Urtreger, A. (1); Noebels, J. L.; Goldner, F. M.; Patrick, J.; Beaudet, A. L. (1)
CORPORATE SOURCE: (1) Mol. Human Genet., Baylor Coll. Med., Houston, TX USA
SOURCE: American Journal of Human Genetics, (1996) Vol. 59, No. 4 SUPPL., pp. A53.
Meeting Info.: 46th Annual Meeting of the American Society of Human Genetics San Francisco, California, USA October 29-November 2, 1996
ISSN: 0002-9297.
DOCUMENT TYPE: Conference
LANGUAGE: English

L41 ANSWER 26 OF 37 BIOSIS COPYRIGHT 1999 BIOSIS
ACCESSION NUMBER: 1995:470010 BIOSIS
DOCUMENT NUMBER: PREV199598484310
TITLE: **Gene** structure and cDNA mutation screening of **alpha-7** neuronal **nicotinic** acetylcholine **receptor** in control and schizophrenic subjects.
AUTHOR(S): Drebing, C. (1); Davis, A.; Hopkins, J.; Barnart, M.; Logel, J.; Freedman, R.; Leonard, S.
CORPORATE SOURCE: (1) Dep. Pharmacology, Univ. Colo. Health Sciences Cent., Denver, CO 80262 USA
SOURCE: Society for Neuroscience Abstracts, (1995) Vol. 21, No. 1-3, pp. 1332.
Meeting Info.: 25th Annual Meeting of the Society for Neuroscience San Diego, California, USA November 11-16, 1995

ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English

L41 ANSWER 27 OF 37 MEDLINE
ACCESSION NUMBER: 96164254 MEDLINE
DOCUMENT NUMBER: 96164254
TITLE: Promoter elements conferring neuron-specific expression of the beta 2-subunit of the neuronal nicotinic acetylcholine receptor studied in vitro and in transgenic mice.
AUTHOR: Bessis A; Salmon A M; Zoli M; Le Nov`ere N; Picciotto M; Changeux J P
CORPORATE SOURCE: UA CNRS D1284, Departement des Biotechnologies, Institut Pasteur 25/28, Paris, France.
SOURCE: NEUROSCIENCE, (1995 Dec) 69 (3) 807-19.
Journal code: NZR. ISSN: 0306-4522.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-X82655; GENBANK-M31433; GENBANK-M90489; GENBANK-M55301; GENBANK-X63509; GENBANK-L11891; GENBANK-X17102
ENTRY MONTH: 199606
AB Several genes encoding subunits of the neuronal nicotinic acetylcholine receptors have been cloned and regulatory elements involved in the transcription of the alpha 2 and **alpha 7**-subunit genes have been described. Yet, the detailed mechanisms governing the neuron-specific transcription and the spatio-temporal expression pattern of these genes remain largely uninvestigated. The beta 2-subunit is the most widely expressed neuronal nicotinic receptor subunit in the nervous system. We have studied the structural and regulatory properties of the 5' sequence of this gene. A fragment of 1163 bp of upstream sequence is sufficient to drive the cell-specific transcription of a reporter gene in both transient transfection assays and in transgenic mice. Deletion analysis and site-directed mutagenesis of this promoter reveal two negative elements and one positive element. The positively-acting sequence includes one functional E-box. One of the repressor elements is located in the transcribed region and is the NRSE/RE1 sequence already described in promoters of neuronal genes. In this paper, we describe the neuron-specific promoter of the gene encoding the neuronal nicotinic acetylcholine receptor beta 2-subunit.

L41 ANSWER 28 OF 37 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 95324936 MEDLINE
DOCUMENT NUMBER: 95324936
TITLE: Cloning and mapping of the mouse **alpha 7** -neuronal nicotinic acetylcholine receptor.
AUTHOR: Orr-Urtreger A; Seldin M F; Baldini A; Beaudet A L
CORPORATE SOURCE: Department of Molecular and Human Genetics, Baylor College of Medicine, Houston, Texas, USA.
CONTRACT NUMBER: HG00734 (NHGRI)
HG00210 (NHGRI)
SOURCE: GENOMICS, (1995 Mar 20) 26 (2) 399-402.
Journal code: GEN. ISSN: 0888-7543.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L37663
ENTRY MONTH: 199510

AB We report the isolation of cDNA clones for the mouse **alpha 7** neuronal nicotinic acetylcholine receptor subunit (gene symbol Acra7), the only nicotinic receptor subunit known to bind alpha-bungarotoxin in mammalian brain. This gene may have relevance to nicotine sensitivity and to some electrophysiologic findings in schizophrenia. The mouse **alpha 7** subunit gene encodes a protein of 502 amino acids with substantial identity to the rat (99.6%), human (92.8%), and chicken (87.5%) amino acid sequences. The **alpha 7** gene was mapped to mouse chromosome 7 near the p locus with the following gene order from proximal to distal: Myod1-3.5 +/- 1.7 cM-Gas2-0.9 cM +/- 0.9 cM-D7Mit70-1.8 +/- 1.2 cM-Acra7-4.4 +/- 1.0 cM-Hras1-ps1/Igf1r/Snrp2a. The human gene was confirmed to map to the homologous region of human chromosome 15q13-q14.

L41 ANSWER 29 OF 37 MEDLINE
ACCESSION NUMBER: 96039222 MEDLINE
DOCUMENT NUMBER: 96039222
TITLE: Stable expression and pharmacological properties of the human **alpha 7** nicotinic acetylcholine receptor.
AUTHOR: Gopalakrishnan M; Buisson B; Touma E; Giordano T; Campbell J E; Hu I C; Donnelly-Roberts D; Arneric S P; Bertrand D; Sullivan J P
CORPORATE SOURCE: Pharmaceutical Products Division, Abbott Laboratories, Abbott Park, IL 60064-3500, USA.
SOURCE: EUROPEAN JOURNAL OF PHARMACOLOGY, (1995 Aug 15) 290 (3) 237-46.
Journal code: EN6. ISSN: 0014-2999.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199602
AB The **alpha 7** neuronal nicotinic acetylcholine receptor subtype forms a Ca(2+)-permeable homooligomeric ion channel sensitive to alpha-bungarotoxin in Xenopus oocytes. In this study, we have stably and functionally expressed the human **alpha 7** cDNA in a mammalian cell line, HEK-293 and examined its pharmacologic properties. [125I] alpha-Bungarotoxin bound to transfected cells with a Kd value of 0.7 nM and a Bmax value of 973 pmoL/mg protein. No specific binding was detected in untransfected cells. Specific binding could be displaced by unlabeled alpha-bungarotoxin (Ki = 0.5 nM) and an excellent correlation was observed between binding affinities of a series of nicotinic cholinergic ligands in transfected cells and those in the human neuroblastoma IMR-32 cell line. Additionally, cell surface expression of **alpha 7** receptors was detected by fluorescein isothiocyanate-conjugated alpha-bungarotoxin in transfected cells. Whole cell currents sensitive to blockade by alpha-bungarotoxin, and with fast kinetics of activation and inactivation, were recorded from transfected cells upon rapid application of (-)-nicotine or acetylcholine with EC50 values of 49 microM and 155 microM respectively. We conclude that the human **alpha 7** subunit when expressed alone can form functional ion channels and that the stably transfected HEK-293 cell line serves as a unique system for studying human **alpha 7** nicotinic receptor function and regulation, and for examining ligand interactions.

L41 ANSWER 30 OF 37 BIOSIS COPYRIGHT 1999 BIOSIS
ACCESSION NUMBER: 1995:477170 BIOSIS
DOCUMENT NUMBER: PREV199598491470
TITLE: The **alpha-7** neuronal **nicotinic**

acetylcholine **receptor** subunit **gene**:
Polymorphism in the human **gene** and generation of hypomorphic mutant mice.
AUTHOR(S): Orr-Urtreger, A. (1); Jiang, Y.-H. (1); Goldner, F. M.; Patrick, J. W.; Beaudet, A. L. (1)
CORPORATE SOURCE: (1) Dep. Mol. Human Genetics, Baylor Coll. Med., Houston, TX USA
SOURCE: American Journal of Human Genetics, (1995) Vol. 57, No. 4 SUPPL., pp. A149.
Meeting Info.: 45th Annual Meeting of the American Society of Human Genetics Minneapolis, Minnesota, USA October 24-28, 1995
ISSN: 0002-9297.
DOCUMENT TYPE: Conference
LANGUAGE: English

L41 ANSWER 31 OF 37 MEDLINE
ACCESSION NUMBER: 95152863 MEDLINE
DOCUMENT NUMBER: 95152863
TITLE: Distribution of nicotinic receptors in the human hippocampus and thalamus.
AUTHOR: Rubboli F; Court J A; Sala C; Morris C; Chini B; Perry E; Clementi F
CORPORATE SOURCE: CNR Centre of Cytopharmacology, Department of Medical Pharmacology, University of Milan, Italy..
SOURCE: EUROPEAN JOURNAL OF NEUROSCIENCE, (1994 Oct 1) 6 (10) 1596-604.
Journal code: BYG. ISSN: 0953-816X.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199505
AB Neuronal nicotinic acetylcholine receptors consist of different subunits, alpha and beta, with different subtype arrangement corresponding to distinct pharmacological and functional properties. The expression of alpha 3, **alpha 7** and beta 2 mRNA in the human brain was studied by in situ hybridization and compared to [3H]nicotine, [3H]cytisine and [125I]alpha-bungarotoxin binding in contiguous sections. The beta 2 probe showed a strong hybridization signal in the granular layer of the dentate gyrus and in the CA2/CA3 region of the hippocampus and in the insular cortex, and a signal of lower intensity in the subicular complex and entorhinal cortex. The alpha 3 probe showed strong hybridization in the dorsomedial, lateral posterior, ventroposteromedial and reticular nuclei of the thalamus, and a weak signal in the hippocampal region and in the entorhinal, insular and cingular cortex. The amount of **alpha 7** mRNA was high at the level of the dentate granular layer and the CA2/CA3 region of the hippocampus, in the caudate nucleus and in the pulvinar and ventroposterolateral nuclei of the thalamus. [3H]Nicotine and [3H]cytisine binding appeared to be identical in anatomical distribution and relative intensity. It was high in the thalamic nuclei, the putamen and in the hippocampal formation in the subicular complex and the stratum lacunosum moleculare. The level of [125I]alpha-bungarotoxin binding was particularly high in the hippocampus and in the pyramidal cells of the CA1 region, but was relatively low in the subicular complex. Our data indicate that in the human brain nicotinic receptor subtypes have discrete distributions, which are in part different from those of other species.

L41 ANSWER 32 OF 37 BIOSIS COPYRIGHT 1999 BIOSIS
ACCESSION NUMBER: 1994:510477 BIOSIS

DOCUMENT NUMBER: PREV199497523477
TITLE: Activity regulates **alpha-7** **nicotinic** ACh **receptor** subunit **gene** expression in rat sympathetic neurons.
AUTHOR(S): De Koninck, P. (1); Cooper, E.
CORPORATE SOURCE: (1) Dep. Biol., McGill Univ., Monteral, PQ H3G 1Y6 Canada
SOURCE: Society for Neuroscience Abstracts, (1994) Vol. 20, No. 1-2, pp. 1131.
Meeting Info.: 24th Annual Meeting of the Society for Neuroscience Miami Beach, Florida, USA November 13-18, 1994
ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English

L41 ANSWER 33 OF 37 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 94245214 MEDLINE
DOCUMENT NUMBER: 94245214
TITLE: Molecular cloning and chromosomal localization of the human **alpha 7**-nicotinic receptor subunit gene (CHRNA7).
AUTHOR: Chini B; Raimond E; Elgoyhen A B; Moralli D; Balzaretti M; Heinemann S
CORPORATE SOURCE: Molecular Neurobiology Laboratory, Salk Institute, La Jolla, California 92037..
CONTRACT NUMBER: NS11549 (NINDS)
SOURCE: GENOMICS, (1994 Jan 15) 19 (2) 379-81.
Journal code: GEN. ISSN: 0888-7543.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-Z23141
ENTRY MONTH: 199408
AB We have isolated cDNA and genomic clones coding for the human **alpha 7** neuronal nicotinic receptor subunit, the major component of brain nicotinic receptors that are blocked by alpha-bungarotoxin. The human **alpha 7** neuronal nicotinic cDNA encodes a mature protein of 479 amino acids that is highly homologous to the rat **alpha 7** neuronal nicotinic subunit (90%). We have mapped the human **alpha 7** -nicotinic receptor subunit gene to chromosome 15, band q14, a region frequently rearranged in patients carrying a bisatellite 15 chromosome, large inv dup (15), whose clinical features include mental retardation and seizures.

L41 ANSWER 34 OF 37 MEDLINE
ACCESSION NUMBER: 94200387 MEDLINE
DOCUMENT NUMBER: 94200387
TITLE: Serotonin release and cell proliferation are under the control of alpha-bungarotoxin-sensitive nicotinic receptors in small-cell lung carcinoma cell lines.
AUTHOR: Codignola A; Tarroni P; Cattaneo M G; Vicentini L M; Clementi F; Sher E
CORPORATE SOURCE: CNR Center of Cytopharmacology, University of Milan, Italy.
SOURCE: FEBS LETTERS, (1994 Apr 11) 342 (3) 286-90.
Journal code: EUH. ISSN: 0014-5793.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199407

AB Neuronal type nicotinic acetylcholine receptors (nAchRs) have recently been identified in small-cell lung carcinoma. We here show that both nicotine and cytisine stimulate [3H]serotonin release in a dose-dependent manner; this effect is antagonized by alpha-bungarotoxin (alpha Bgtx) and alpha-conotoxin MI (alpha Ctx). Nicotine and cytisine stimulate in vitro SCLC proliferation and this effect is completely antagonized by both alpha Bgtx and alpha Ctx. By PCR analysis, we demonstrate the presence in SCLC of both the **alpha 7** and the beta 2 nAchR subunits mRNA. These data show that nAchRs play an important role in the biology of SCLC, and that alpha Bgtx-sensitive receptors of the **alpha 7** subtype are crucially involved in both the secretagogue and mitogenic effects of nicotinic agonists.

L41 ANSWER 35 OF 37 BIOSIS COPYRIGHT 1999 BIOSIS
ACCESSION NUMBER: 1994:524980 BIOSIS
DOCUMENT NUMBER: PREV199497537980
TITLE: The neuronal **nicotinic** acetylcholine **receptor alpha-7** subunit **gene**: Cloning, mapping, structure, and targeting in mouse.
AUTHOR(S): Orr-Urtreger, A. (1); Seldin, M. F.; Baldin, A. (1); Beaudet, A. L. (1)
CORPORATE SOURCE: (1) Dep. Mol. Human Genetics, Baylor Coll. Med., Houston, TX USA
SOURCE: American Journal of Human Genetics, (1994) Vol. 55, No. 3 SUPPL., pp. A136.
Meeting Info.: 44th Annual Meeting of the American Society of Human Genetics Montreal, Quebec, Canada October 18-22, 1994
ISSN: 0002-9297.
DOCUMENT TYPE: Conference
LANGUAGE: English

L41 ANSWER 36 OF 37 BIOSIS COPYRIGHT 1999 BIOSIS
ACCESSION NUMBER: 1994:5389 BIOSIS
DOCUMENT NUMBER: PREV199497018389
TITLE: A strain comparison of **alpha 7 nicotinic receptor gene** expression in mouse brain.
AUTHOR(S): Pauly, J. R. (1); Grun, E. U.; Gross, S. D.; Marks, M. J.; Collins, A. C.
CORPORATE SOURCE: (1) Dep. Pharmacol., Medical College Georgia, Augusta, GA 30912 USA
SOURCE: Society for Neuroscience Abstracts, (1993) Vol. 19, No. 1-3, pp. 465.
Meeting Info.: 23rd Annual Meeting of the Society for Neuroscience Washington, D.C., USA November 7-12, 1993
ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English

L41 ANSWER 37 OF 37 BIOSIS COPYRIGHT 1999 BIOSIS
ACCESSION NUMBER: 1993:77374 BIOSIS
DOCUMENT NUMBER: PREV199395041874
TITLE: Neuronal specificity of the alpha-7 nicotinic acetylcholine receptor promoter develops during morphogenesis of the central nervous system.
AUTHOR(S): Matter-Sadzinski, Lidia; Hernandez, Maria-Clemencia; Roztocil, Tomas; Ballivet, Marc; Matter, Jean-Marc
CORPORATE SOURCE: Dep. Biochem., Sci. II, Univ. Geneva, 1211 Geneva 4 Switzerland

SOURCE: EMBO (European Molecular Biology Organization) Journal, (1992) Vol. 11, No. 12, pp. 4529-4538.
ISSN: 0261-4189.
DOCUMENT TYPE: Article
LANGUAGE: English

AB A transient transfection assay has been developed to analyse promoter activity in neuronal cells freshly dissociated from the chick central nervous system. The assay enabled us to identify cis-acting regulatory elements within the 5'-flanking region of the **alpha-7 nicotinic** acetylcholine **receptor gene**. In differential retina, regulatory elements direct reporter **gene** expression to a small subset of neurons which has been identified as ganglion cells, i.e. to the population of neurons in which alpha-7 transcripts were localized by in situ hybridization. However, these promoter elements exhibit ubiquitous activity in undifferentiated neural cells and in mesodermal stem cells. Our study supports the idea that alpha-7 regulatory elements acquire their neuronal specificity in the course of embryogenesis.

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